```
=> s cox-2 () inhibit?
          13814 COX
              2 COXES
          13816 COX
                (COX OR COXES)
        8066459 2
           6586 COX-2
                (COX(W)2)
        1660366 INHIBIT?
L1         2615 COX-2 (W) INHIBIT?

=> s l1 and inflammat?
         179141 INFLAMMAT?
L2         1536 L1 AND INFLAMMAT?

=> s l2 and review/dt
        1734424 REVIEW/DT
L3          434 L2 AND REVIEW/DT

=> s l1 and inflammat? () disorder?
         179141 INFLAMMAT?
         374069 DISORDER?
           1796 INFLAMMAT? (W) DISORDER?
L4           22 L1 AND INFLAMMAT? (W) DISORDER?

=> s l4 and review/dt
        1734424 REVIEW/DT
L5            6 L4 AND REVIEW/DT

=> d l5, ibib abs, 1-6

L5   ANSWER 1 OF 6  HCAPLUS  COPYRIGHT 2004 ACS on STN
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Full Text | Citing References

ACCESSION NUMBER: 2003:969623 HCAPLUS
DOCUMENT NUMBER: 140:399057
TITLE: The prospective use of **COX-2 inhibitors** for the treatment of temporomandibular joint **inflammatory disorders**
AUTHOR(S): Kerins, C. A.; Spears, R.; Bellinger, L. L.; Hutchins, B.
CORPORATE SOURCE: Department of Biomedical Sciences, Texas A and M University System Health Science Center, Baylor College of Dentistry, Dallas, TX, 75246, USA
SOURCE: International Journal of Immunopathology and Pharmacology (2003), 16(2, Suppl.), 1-9
CODEN: IJIPE4; ISSN: 0394-6320
PUBLISHER: Biolife s.a.s.
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English

AB A review. Development of a new class of drugs designed to selectively inhibit the inducible cyclooxygenase isoenzyme, COX-2, was initially prescribed for individuals diagnosed with osteoarthritis or rheumatoid arthritis. Although these **inflammatory disorders** are more typically related to the joints of the knee, ankle, or hand, the temporomandibular joint (TMJ) plays a special role due to its involvement in our normal day-to-day activities of eating and communicating. The TMJ, unlike most of the other joints, contains some unique morphol. characteristics that support various **inflammatory disorders**. An overview of these characteristics and the prospective use of the **COX-2 inhibitors** for

temporomandibular joint inflammation are presented.
REFERENCE COUNT: 80 THERE ARE 80 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 2 OF 6 HCAPLUS COPYRIGHT 2004 ACS on STN
Full Text | Citing References

ACCESSION NUMBER: 2003:919785 HCAPLUS
DOCUMENT NUMBER: 139:374127
TITLE: Balancing gastroprotection and cardioprotection with selective cyclo-oxygenase-2 inhibitors: Clinical implications
AUTHOR(S): Meagher, Emma A.
CORPORATE SOURCE: Department of Medicine and Center for Experimental Therapeutics, University of Pennsylvania School of Medicine, Philadelphia, USA
SOURCE: Drug Safety (2003), 26(13), 913-924
CODEN: DRSAEA; ISSN: 0114-5916
PUBLISHER: Adis International Ltd.
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English
AB A review. NSAIDs have been the mainstay of treatment in the management of pain and inflammation assocd. with chronic **inflammatory disorders**. They are effective. However, complications arising from chronic NSAID use are common and are primarily due to gastrointestinal (GI) toxicity in the form of gastritis, peptic erosions and ulceration and GI bleeds. GI toxicity has been attributed to the blockade of the cyclo-oxygenase (COX)-1-mediated generation of the cytoprotective prostanoids, such as prostaglandin (PG) E2 and PGI2 (prostacyclin). More recently, selective **COX-2 inhibitors** ('coxibs') were designed to inhibit the prodn. of COX-2-dependent inflammatory prostanoids and to leave intact the cytoprotective COX-1 products. The coxibs, while exhibiting similar efficacy to traditional NSAIDs in controlled clin. trials of their efficacy in chronic inflammatory conditions, such as osteoarthritis and rheumatoid arthritis, have been assocd. with a reduced incidence of surrogate or actual indexes of GI toxicity. However, concerns regarding cardiovascular safety in high-risk patients have evolved. These concerns were driven initially by the concept that inhibition of COX-2-derived endothelial PGI2 without concomitant inhibition of platelet thromboxane A2 would result in increased cardiovascular risk. This was borne out in the Vioxx Gastrointestinal Outcomes Research study of rofecoxib, but not demonstrated in the Celecoxib Long Term Arthritis Safety Study trial. Further elucidation of the relative roles of COX-1- and COX-2-generated prostanoids has enabled a greater understanding of the biol. of these pathways. However, it is still not completely clear how this understanding may be appropriately translated into clin. medicine.
REFERENCE COUNT: 70 THERE ARE 70 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 3 OF 6 HCAPLUS COPYRIGHT 2004 ACS on STN
Full Text | Citing References

ACCESSION NUMBER: 2003:533041 HCAPLUS
DOCUMENT NUMBER: 139:190463
TITLE: Role of cyclooxygenase-2 inhibitors in combination with radiation therapy in lung cancer
AUTHOR(S): Liao, Zhongxing; Komaki, Ritsuko; Mason, Kathryn A.; Milas, Luka
CORPORATE SOURCE: Department of Radiation Oncology, University of Texas M. D. Anderson Cancer Center, Houston, USA

SOURCE: Clinical Lung Cancer (2003), 4(6), 356-365
CODEN: CLCLCA; ISSN: 1525-7304
PUBLISHER: Cancer Information Group
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English
AB A review. Cyclooxygenase-2 (COX-2) is an enzyme involved in prostaglandin prodn. in pathol. states such as **inflammatory disorders** and cancer. The enzyme is often overexpressed in premalignant lesions and cancer of the lung. Overexpression of COX-2 in lung cancer is assocd. with more aggressive biol. tumor behavior and adverse patient outcome. In preclin. studies, inhibition of this enzyme with selective **COX-2 inhibitors** enhances tumor response to radiation and chemotherapeutic agents. These findings have been rapidly advanced to clin. oncol. Clin. trials of the combination of selective **COX-2 inhibitors** with radiation therapy, chemotherapy, or both in patients with lung cancer have been initiated and some preliminary results are available. In this review, we describe the relation between overexpression of COX-2 and lung cancer, the antitumor effect of selective **COX-2 inhibitors**, discuss the rationale for using selective **COX-2 inhibitors** combined with radiation therapy and chemotherapy, and summarize current clin. protocols and initial findings.
REFERENCE COUNT: 114 THERE ARE 114 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 4 OF 6 HCAPLUS COPYRIGHT 2004 ACS on STN
Full Text | Citing References
ACCESSION NUMBER: 2002:929912 HCAPLUS
DOCUMENT NUMBER: 139:78127
TITLE: Selective **COX-2 inhibitors** - a review
AUTHOR(S): Joshi, U. J.; Sangshetti, J.
CORPORATE SOURCE: Dep. of Pharm. Chem., Prin. K.M. Kundnani Coll. of Pharm., Mumbai, 400018, India
SOURCE: Indian Drugs (2002), 39(7), 355-359
CODEN: INDRBA; ISSN: 0019-462X
PUBLISHER: Indian Drug Manufacturers' Association
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English
AB A review. The article discusses the mode of action and various chem. classes of selective **COX-2 inhibitors** which constitute a major advance in the treatment of **inflammatory disorders**.
REFERENCE COUNT: 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 5 OF 6 HCAPLUS COPYRIGHT 2004 ACS on STN
Full Text | Citing References
ACCESSION NUMBER: 2001:725965 HCAPLUS
DOCUMENT NUMBER: 136:63482
TITLE: Changes in ulcerogenic response to non-steroidal anti-inflammatory drugs (NSAIDS) in adjuvant arthritic rats
AUTHOR(S): Kato, Shinichi
CORPORATE SOURCE: Kyoto Pharmaceutical Univ., Kyoto, 607-8414, Japan
SOURCE: Yakugaku Zasshi (2001), 121(10), 743-751
CODEN: YKKZAJ; ISSN: 0031-6903
PUBLISHER: Pharmaceutical Society of Japan
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: Japanese
AB A review with refs. Gastroenteropathy is the most common among patients

who use non-steroidal anti-inflammatory drugs (NSAIDs) for the treatment of **inflammatory disorders**. It is known that rheumatoid arthritic (RA) patients are more susceptible to NSAID-induced gastropathy than other NSAID users. This article reviewed our recent studies concerning the influence of arthritis on gastric mucosal integrity in adjuvant-induced arthritic rats. The gastric mucosal lesions induced by indomethacin, one of conventional NSAIDs, were markedly aggravated in arthritic rats. Likewise, the healing of chronic gastric ulcers induced by thermal cauterization was significantly delayed in arthritic rats. The underlying mechanism of these phenomena obsd. in arthritic rats may be attributable to the enhancement of iNOS/NO pathway in the former and the less expression of various growth factors in the ulcerated mucosa, such as basic fibroblast growth factors (bFGF) or insulin-like growth factors (IGF-1) in the latter. In addn., we recently found that cyclooxygenase-2 (COX-2) selective inhibitors, such as rofecoxib or celecoxib, induced apparent gastric lesions in arthritic rats, suggesting that a caution should be paid on the use of COX-2 selective inhibition in RA patients.

L5 ANSWER 6 OF 6 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 1998:404547 HCAPLUS
DOCUMENT NUMBER: 129:173761
TITLE: COX-2, TNF-α and apoptosis: newer strategies in **inflammatory disorders**
AUTHOR(S): Kulkarni, S. K.; Varghese, Navin P.
CORPORATE SOURCE: Pharmacology Division, University Institute of Pharmaceutical Sciences, Panjab University, Chandigarh, 160 014, India
SOURCE: Indian Drugs (1998), 35(5), 245-260
CODEN: INDRBA; ISSN: 0019-462X
PUBLISHER: Indian Drug Manufacturers' Association
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English
AB A review, with 73 refs. Inflammatory conditions related to rheumatoid arthritis, injury and infection necessitates the need for the use of nonsteroidal antiinflammatory drugs (NSAIDs) to curb these ailments. There have been high levels of concern regarding their use since extensive and indiscriminate use of NSAIDs results in toxicity. The need for an overall therapeutic benefit with no or reduced toxicity has envisaged the need for investigating newer sites of antiinflammatory activity. This article highlights the development of selective **COX-2 inhibitors**, and newer concepts of antiTNF-α therapy and induction of apoptosis as potential strategies in combating inflammation.
REFERENCE COUNT: 73 THERE ARE 73 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d his

(FILE 'HOME' ENTERED AT 10:48:57 ON 15 JUN 2004)

FILE 'REGISTRY' ENTERED AT 10:49:17 ON 15 JUN 2004

FILE 'HCAPLUS' ENTERED AT 10:49:39 ON 15 JUN 2004
L1 2615 S COX-2 () INHIBIT?
L2 1536 S L1 AND INFLAMMAT?
L3 434 S L2 AND REVIEW/DT
L4 22 S L1 AND INFLAMMAT? () DISORDER?
L5 6 S L4 AND REVIEW/DT

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=> s l1 and ulcerat? () colit?
          9973 ULCERAT?
          7724 COLIT?
          4735 ULCERAT? (W) COLIT?
L6          41 L1 AND ULCERAT? (W) COLIT?

=> s l6 and review/dt
       1734424 REVIEW/DT
L7           1 L6 AND REVIEW/DT

=> d l7, ibib abs, 1

L7   ANSWER 1 OF 1  HCAPLUS  COPYRIGHT 2004 ACS on STN
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Full Text | Citing References

ACCESSION NUMBER: 2002:661516 HCAPLUS
DOCUMENT NUMBER: 138:378294
TITLE: Nonsteroidal anti-inflammatory drugs and inflammatory bowel disease: current perspectives
AUTHOR(S): Cipolla, Giovanna; Crema, Francesca; Sacco, Stefano; Moro, Elisabetta; De Ponti, Fabrizio; Frigo, Gianmario
CORPORATE SOURCE: Department of Internal Medicine and Therapeutics, University of Pavia, Pavia, 27100, Italy
SOURCE: Pharmacological Research (2002), 46(1), 1-6
CODEN: PHMREP; ISSN: 1043-6618
PUBLISHER: Elsevier Science Ltd.
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English

AB A review. Mechanisms underlying the gastric toxicity of nonsteroidal anti-inflammatory drugs (NSAIDs) have been extensively investigated, whereas those leading to intestinal damage are not completely understood. Several hypotheses have been put forward on the pathophysiol. of intestinal damage by NSAIDs: enhanced intestinal permeability, inhibition of cyclooxygenase (COX), enterohepatic recirculation, and formation of adducts. The effects of COX-2 selective inhibitors, which appear to have better gastric tolerability when compared to nonselective NSAIDs, on normal and inflamed intestinal mucosa (as in Crohn's disease or **ulcerative colitis**) are still largely unexplored. If **COX-2 inhibition** plays a key role in suppressing the inflammatory process, recent evidence suggests that COX-2 products are involved in maintaining the integrity of intestinal mucosa, in the healing of gastrointestinal ulcers and in the modulation of inflammatory bowel disease (IBD). Animal models of intestinal inflammation have so far yielded conflicting results on the effects of COX-2 selective inhibitors on the intestinal mucosa. It is now clear that NSAIDs do not act through cyclooxygenase inhibition, but also have different targets such as nuclear factor-κB and/or peroxisome proliferator-activated receptors γ. The peculiar pharmacol. profile of each compd. may help to explain the different impact of each NSAID on the inflammatory process and on IBD. Notably, the salicylic acid deriv. 5-ASA is widely used in the treatment of IBD and is believed to act through nuclear factor-κB inhibition. Although the use of COX-2 selective inhibitors remains contraindicated in patients with IBD, studying their effects on intestinal mucosa may offer new insights into their subcellulars mechanisms of action and open new avenues for the development of novel therapies for IBD.

REFERENCE COUNT: 56 THERE ARE 56 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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     (FILE 'HOME' ENTERED AT 10:48:57 ON 15 JUN 2004)

     FILE 'REGISTRY' ENTERED AT 10:49:17 ON 15 JUN 2004

     FILE 'HCAPLUS' ENTERED AT 10:49:39 ON 15 JUN 2004
L1          2615 S COX-2 () INHIBIT?
L2          1536 S L1 AND INFLAMMAT?
L3           434 S L2 AND REVIEW/DT
L4            22 S L1 AND INFLAMMAT? () DISORDER?
L5             6 S L4 AND REVIEW/DT
L6            41 S L1 AND ULCERAT? () COLIT?
L7             1 S L6 AND REVIEW/DT

=> s l1 and asthma?
         25341 ASTHMA?
L8          72 L1 AND ASTHMA?

=> s l8 and review/dt
       1734424 REVIEW/DT
L9          12 L8 AND REVIEW/DT

=> d l9, ibib abs, 1-12

L9   ANSWER 1 OF 12  HCAPLUS  COPYRIGHT 2004 ACS on STN
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Full Text | Citing References

ACCESSION NUMBER: 2003:969624 HCAPLUS
DOCUMENT NUMBER: 140:12337
TITLE: COX-2 specific inhibitors in NSAID-intolerant patients
AUTHOR(S): Picado, C.
CORPORATE SOURCE: Servei de Pneumologia i Allergia Respiratoria, Institut Clinic de Panumologia i Cirurgia Toracia, Hospital Clinic, Department de Medicina, Universitat de Barcelona, Barcelona, 08036, Spain
SOURCE: International Journal of Immunopathology and Pharmacology (2003), 16(2, Suppl.), 11-16
CODEN: IJIPE4; ISSN: 0394-6320
PUBLISHER: Biolife s.a.s.
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English

AB A review. Most adverse NSAID-induced respiratory and skin reactions appear to be pptd. by the inhibition of cyclooxygenase-1 (COX-1); this in turn activates the lypoxygenase pathway, which eventually increases the release of cysteinyl leukotrienes (Cys-LTs). Recent studies have reported that patients with NSAID-induced **asthma** have a low prodn. of PGE2 in respiratory epithelial cells, bronchial fibroblast and peripheral blood cells. Low prodn. of PGE2 may be due to an insufficient cyclooxygenase-2 (COX-2) expression in the inflammatory response underlying **asthma**. Since PGE2 administered by inhalation inhibits NSAID-induced bronchoconstriction and the parallel increase in Cys-LTs release, a reduced PGE2 synthesis may render NSAID-patients more susceptible to the COX-1 inhibitory effects of NSAIDs. Recent studies have shown that selective **COX-2 inhibitors** (rofecoxib and celecoxib), unlike COX-1 inhibitors, are very well tolerated by NSAID-sensitive patients and do not elicit increased Cyst-LTs prodn. However, these drugs can still can ppt. cutaneous reactions in a significant proportion of patients with skin reactions to NSAID. The heterogeneity of the NSAID-intolerance syndrome

suggests that subjects who do not tolerate NSAID can use coxibs only after first having been exposed to the drug under the supervision of a specialist with experience in these procedures.

REFERENCE COUNT: 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 2 OF 12 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2003:839655 HCAPLUS
DOCUMENT NUMBER: 139:357854
TITLE: Safety of **COX-2 inhibitors** in **asthma** patients with aspirin hypersensitivity
AUTHOR(S): West, Patricia M.; Fernandez, Cristina
CORPORATE SOURCE: Department of Pharmacy Practice, College of Pharmacy, University of Illinois at Chicago, Chicago, IL, USA
SOURCE: Annals of Pharmacotherapy (2003), 37(10), 1497-1501
CODEN: APHRER; ISSN: 1060-0280
PUBLISHER: Harvey Whitney Books Co.
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English

AB A review. Objective: To review the safety of cyclooxygenase-2 (**COX-2**) **inhibitors** in **asthma** patients with aspirin hypersensitivity. Data Sources: Clin. studies were identified using MEDLINE (1966-Sept. 2002). Key search terms included cyclooxygenase inhibitors, aspirin, **asthma**, and hypersensitivity. English-language articles were identified and included. Refs. from the identified articles were also reviewed. Data Synthesis: The literature provides information regarding the safety of **COX-2 inhibitors** in **asthma** patients with aspirin-exacerbated respiratory disease (AERD). The mechanism of AERD involves inhibition of cyclooxygenase, particularly COX-1. Inhibition of COX-1 causes an increased prodn. of certain inflammatory mediators, which results in the reactions seen with AERD. Considering this mechanism, **COX-2 inhibitors** may be an alternative to aspirin or nonsteroidal antiinflammatory drugs (NSAIDs) in a patient with AERD. This article analyzes 4 studies to evaluate the safety of **COX-2 inhibitors** in this population. Results: The 4 studies evaluated included a total of 172 patients with AERD. All patients included demonstrated intolerance to aspirin or NSAIDs and tolerated the selective **COX-2 inhibitor** administered. Conclusions: **COX-2 inhibitors** provide a potentially safe alternative for treatment of inflammatory conditions in patients with AERD.

REFERENCE COUNT: 14 THERE ARE 14 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 3 OF 12 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2003:425591 HCAPLUS
DOCUMENT NUMBER: 139:373869
TITLE: Aspirin-induced **asthma**: Advances in pathogenesis, diagnosis, and management
AUTHOR(S): Szczeklik, Andrew; Stevenson, Donald D.
CORPORATE SOURCE: Department of Medicine, Jagellonian University, Krakow, Pol.
SOURCE: Journal of Allergy and Clinical Immunology (2003), 111(5), 913-921
CODEN: JACIBY; ISSN: 0091-6749
PUBLISHER: Mosby, Inc.
DOCUMENT TYPE: Journal; **General Review**

LANGUAGE: English

AB A review. In some **asthmatic** individuals, aspirin and other nonsteroidal anti-inflammatory drugs (NSAIDs) that inhibit cyclooxygenase 1 (COX-1) exacerbate the condition. This distinct clin. syndrome, called aspirin-induced **asthma** (AIA), is characterized by an eosinophilic rhinosinusitis, nasal polyposis, aspirin sensitivity, and **asthma**. There is no in vitro test for the disorder, and diagnosis can be established only by provocation challenges with aspirin or NSAIDs. Recent major advances in the mol. biol. of eicosanoids, exemplified by the cloning of 2 cysteinyl leukotriene receptors and the discovery of a whole family of cyclooxygenase enzymes, offer new insights into mechanisms operating in AIA. The disease runs a protracted course even if COX-1 inhibitors are avoided, and the course is often severe, many patients requiring systemic corticosteroids to control their sinusitis and **asthma**. Aspirin and NSAIDs should be avoided, but highly specific **COX-2 inhibitors**, known as coxibs, are well tolerated and can be safely used. Aspirin desensitization, followed by daily aspirin treatment, is a valuable therapeutic option in most patients with AIA, particularly those with recurrent nasal polyposis or overdependence on systemic corticosteroids.

REFERENCE COUNT: 102 THERE ARE 102 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 4 OF 12 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2002:932690 HCAPLUS
DOCUMENT NUMBER: 138:361994
TITLE: Diagnosis, prevention, and treatment of aspirin-induced **asthma** and rhinitis
AUTHOR(S): Bochenek, G.; Banska, K.; Szabo, Z.; Nizankowska, E.; Szczeklik, A.
CORPORATE SOURCE: Department of Medicine, Jagiellonian University School of Medicine, Krakow, Pol.
SOURCE: Current Drug Targets: Inflammation & Allergy (2002), 1(1), 1-11
CODEN: CDTICU; ISSN: 1568-010X
PUBLISHER: Bentham Science Publishers Ltd.
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English

AB A review. Bronchial **asthma** is not a homogeneous disease. Several variants of **asthma** can be distinguished. One of them is aspirin-induced **asthma**. In this distinct clin. syndrome, aspirin and most other nonsteroidal anti-inflammatory drugs that inhibit cyclooxygenase-1 ppt. rhinitis and **asthma** attacks. This type of **asthma** affects 5-10% of adult **asthmatics**, but remains largely underdiagnosed. The natural history of aspirin-induced **asthma** (AIA) was described, based on an extensive pan-European survey. Aspirin provocation tests with improved diagnostic accuracy were developed, although no in-vitro tests was found to be of diagnostic value. Recent interest in AIA was stirred by the finding of alterations in arachidonate metabolic pathways, leading to cysteinyl-leukotriene overprodn. LTC4 synthase is overexpressed in bronchi and its mRNA is upregulated in peripheral blood eosinophils. The gene coding for LTC4 synthase exists in 2 common alleles, 1 of which appears to be assocd. with a severe, steroid-dependent type of **asthma**. New highly specific **COX-2 inhibitors** appear to be a safe alternative for patients with aspirin-induced **asthma**.

REFERENCE COUNT: 141 THERE ARE 141 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 5 OF 12 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2002:136698 HCAPLUS
DOCUMENT NUMBER: 136:395185
TITLE: Non steroidal anti-inflammatory and anti-allergy agents
AUTHOR(S): Kontogiorgis, C. A.; Hadjipavlou-Litina, D. J.
CORPORATE SOURCE: Department of Pharmaceutical Chemistry, School of Pharmacy, Aristotelian University of Thessaloniki, Thessaloniki, 54006, Greece
SOURCE: Current Medicinal Chemistry (2002), 9(1), 89-98
CODEN: CMCHE7; ISSN: 0929-8673
PUBLISHER: Bentham Science Publishers
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English

GOT

AB A review. Non steroidal anti-inflammatory drugs (NSAIDs) are among the most commonly used for inflammation therapy. The major drawback in using the NSAIDs is in their tendency to cause gastrointestinal toxicity. Since the roles of arachidonic acid (A.A) metabolites, as leukotrienes (Lts), prostaglandins (PGs) and thromboxanes (TXA2) as mediators of the inflammatory reaction were clarified, much effort has been made to develop inhibitors of the prodn. of these chem. mediators as anti-inflammatory agents. These mediators also play important roles in some inflammatory or allergic diseases, acting either alone or in combination and inhibitors of 5-lipoxygenase (5-LOX) and/or cyclooxygenase isoforms 1,2 (COX-1,2) may be useful for the treatment of **asthma**, psoriasis and rheumatoid arthritis. Leukotrienes, the products of 5-LOX metab. have been assocd. with immediate hypersensitivity reactions, anaphylaxis and **asthma**. In addn., active oxygen species (AOS) including superoxide anion (O2-), hydrogen peroxide, hydroxyl radical and ferric radical, mediate cell damage in a variety of pathophysiol. conditions and are responsible for oxidative injury of enzymes, lipid membranes and DNA in living cells and tissues. Prostaglandins and leukotrienes in the arachidonate pathway linked with lipid peroxidn. may amplify the oxidative damage. Nitric oxide (NO) plays also a role as an effector in inflammation, since PG and NO thought to be important in maintaining mucosal integrity. Dual or selective inhibitors, specific receptor antagonists, AOS scavengers, and NO donors have been under development for therapeutic application. Several classes of inhibitors have been identified and at least 12 major chem. series are known to affect PGs prodn. directly. In this review, we account on our research work concerning NSAIDs combined with a ref. of the recent literature.

REFERENCE COUNT: 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 6 OF 12 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2001:605206 HCAPLUS
DOCUMENT NUMBER: 136:79046
TITLE: Aspirin, nonsteroidal anti-inflammatory drugs, and preservatives as causes for severe **asthma**
AUTHOR(S): Stevenson, Donald D.
CORPORATE SOURCE: Division of Allergy, Asthma and Immunology, Scripps Clinic and the Scripps Research Institute, La Jolla, CA, USA
SOURCE: Lung Biology in Health and Disease (2001), 159(Severe Asthma), 361-387

CODEN: LBHDD7; ISSN: 0362-3181
PUBLISHER: Marcel Dekker, Inc.
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English
AB A review with refs. discusses the clin. features of aspirin-sensitive **asthma** (ASA) respiratory disease and methods for diagnosing ASA sensitivity. It also covers the prevalence of ASA; cross-reactions with nonsteroidal anti-inflammatory drugs (NSAIDs); lack of cross-reactions with cyclooxygenase-2 (**COX-2**) **inhibiting** NSAIDs, as well as with other drugs and chems.; the phenomenon of ASA desensitization; and treatment. Comments regarding the severity of **asthma** in ASA-sensitive **asthmatics** are focused in two areas, i.e., respiratory reactions and aspirin respiratory disease.
REFERENCE COUNT: 107 THERE ARE 107 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 7 OF 12 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2001:554482 HCAPLUS
DOCUMENT NUMBER: 136:272445
TITLE: The pharmacological profile of ML3000: a new pyrrolizine derivative inhibiting the enzymes cyclo-oxygenase and 5-lipoxygenase
AUTHOR(S): Tries, S.; Laufer, S.
CORPORATE SOURCE: R&D Division, Merckle GmbH, Blaubeuren, 7, 89143, Germany
SOURCE: Inflammopharmacology (2001), 9(1-2), 113-124 Gut
CODEN: IAOAES; ISSN: 0925-4692
PUBLISHER: VSP BV
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English
AB A review with refs. Since the discovery of aspirin about one century ago, many non-steroidal anti-inflammatory drugs (NSAIDs) have been used for the treatment of inflammatory states and pain. While the NSAIDs are generally safe and effective, common side effects frequently limit therapy. Typical mechanism-based side effects are gastrointestinal (GI)-related, ranging from GI upset and intolerance to ulceration and bleeding after long-term therapy. In order to overcome these side effects several strategies have been followed, among them the development of selective **COX-2 inhibitors**. Our strategy to find compds. that are active on the one hand and tolerated by the GI tract on the other hand, is based on the shunt to leukotrienes. This theory is founded upon the fact that NSAIDs, while inhibiting the cyclooxygenase branch of the arachidonic acid cascade, are able to increase the 5-lipoxygenase (5-LOX) branch of arachidonic acid metab. This shunt to the 5-LOX side leads to the increase in chemotactic LTB4 and vasoconstrictive peptidoleukotrienes, the contributory effects of which to gastrointestinal disorders are widely accepted. Therefore, the design of anti-inflammatory compds. with 5-LOX inhibitory effects seems reasonable. With the compd. ML3000, this theory has gained further evidence. ML3000 is an anti-inflammatory compd. with potent activity in various animal expts. that represent models for acute and chronic inflammation, pain, fever and **asthma**. It is a balanced inhibitor of the enzymes 5-LOX and COX-1/2 in the submicromolar range. The compd. demonstrates excellent gastrointestinal tolerance in various animal species. The preclin. profile of ML3000, which is currently in Phase III clin. development, is presented in this publication.
REFERENCE COUNT: 11 THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 8 OF 12 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2001:554475 HCAPLUS
DOCUMENT NUMBER: 135:313019
TITLE: Current issues on the safety of non-prescription NSAIDs
AUTHOR(S): Volans, Glyn
CORPORATE SOURCE: Medical Toxicology Unit, Guy's and St. Thomas' Hospital Trust and King's College, London, SE14 5ER, UK
SOURCE: Inflammopharmacology (2001), 9(1-2), 43-49
CODEN: IAOAES; ISSN: 0925-4692
PUBLISHER: VSP BV
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English

AB A review with refs. There is a continuing need to monitor the safety of non-prescription (OTC) NSAIDs in order to better define known adverse drug reactions; to consider potential drug interactions and to assess the case for further OTC transfers. Recent reviews at the Medical Toxicol. Unit have therefore included: (1) the potential of NSAIDs to induce **asthma** with a view to producing guidelines for safe usage; (2) the possibility of interactions between NSAIDs and alc.; (3) the safety of **COX-2 inhibitors** in overdosage.

REFERENCE COUNT: 11 THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 9 OF 12 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2001:411272 HCAPLUS
DOCUMENT NUMBER: 136:240847
TITLE: Anti-inflammatory drugs: new multitarget compounds to face an old problem. The dual inhibition concept
AUTHOR(S): Celotti, Fabio; Laufer, Stefan
CORPORATE SOURCE: Institute of Endocrinology, University of Milano, Italy
SOURCE: Pharmacological Research (2001), 43(5), 429-436
CODEN: PHMREP; ISSN: 1043-6618
PUBLISHER: Academic Press
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English

AB A review. In this short review we have tried to focus on some new relevant aspects of the pharmacol. control of inflammation. The clin. availability of new drugs able to produce a selective inhibition of type 2 cyclooxygenase (COX-2), the enzyme thought to be mainly responsible for generating arachidonic-acid-derived inflammatory mediators, has been the origin of much hope. However, expectations of having an effective and completely safe non-steroidal anti-inflammatory drug (NSAID) have been only partially fulfilled. Emerging information has challenged some aspects of the original hypothesis indicating COX-2 as devoid of 'housekeeping' physiol. functions. Moreover, the recently available clin. studies have indicated only a relatively small improvement in the tolerability of the newer 'selective' **COX-2 inhibitors** over the classical COX-1/COX-2 mixed type NSAIDs. The new appreciation of the role of other arachidonic acid derivs., the leukotrienes (LTS), in producing and maintaining inflammation has generated considerable interest in drugs able to block LTS receptors or to produce a selective inhibition of 5-lipoxygenase (5-LO), the initial key enzyme of the leukotriene pathway.

These drugs are now included among the effective therapies of **asthma** but appear, in the few clin. studies performed, to be an insufficient single therapeutic approach in other inflammatory diseases. Drugs able to block equally well both COX and 5-LO metabolic pathways (dual inhibitors) have been developed and exptl. evaluated in the last few years, but none are available on the market yet. The pharmacol. rationale at the basis of their development is strong, and animal studies are indicative of a wide range of anti-inflammatory activity. What appears most impressive from the available studies on dual inhibitors is their almost complete lack of gastric toxicity, the most troublesome side effect of NSAIDs. The mechanism of the gastric-sparing properties of these drugs is not yet completely understood; however, it appears that leukotrienes significantly contribute to gastric epithelial injury particularly when these compds. represent the major arachidonic acid derivs. present in the gastric mucosa after inhibition of prostanoid prodn. (c) 2001 The Italian Pharmacological Society.

REFERENCE COUNT: 101 THERE ARE 101 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 10 OF 12 HCAPLUS COPYRIGHT 2004 ACS on STN

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ACCESSION NUMBER: 2001:400485 HCAPLUS
DOCUMENT NUMBER: 136:160669
TITLE: Recent progress in aspirin-induced **asthma**
AUTHOR(S): Sakakibara, Hiroki
CORPORATE SOURCE: Department of Allergy and Internal Medicine, Fujita Health and Hygiene University, Japan
SOURCE: Annual Review Kokyuki (2000) 82-92
CODEN: ARKNC8
PUBLISHER: Chugai Igakusha
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: Japanese

AB A review. Aspirin-induced **asthma** (AIA) is a distinct clin. syndrome in which bronchoconstrictive responses to nonsteroidal anti-inflammatory drugs (NSAIDs) can be predicted on the basis of their in vitro activity as inhibitors of cyclooxygenase, i.e. AIA is assocd. with alterations in arachidonate metab. In this review, several explanations are presented including peptidoleukotrienes overprodn., overexpression of leukotriene C4 (LTC4) synthase in bronchial cells, 5-lipoxygenase and LTC4 synthase gene promoter polymorphism, PGE2 dependency, role of the mast cells, and specific **COX-2 inhibitors**.

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ACCESSION NUMBER: 2000:598134 HCAPLUS
DOCUMENT NUMBER: 134:50880
TITLE: New selective **COX-2 inhibitors**
AUTHOR(S): Kam, P. C. A.; Power, I.
CORPORATE SOURCE: Department of Anaesthesia and Pain Management, Royal North Shore Hospital, University of Sydney, St Leonards, 2065, Australia
SOURCE: Pain Reviews (2000), 7(1), 3-13
CODEN: PAREFV; ISSN: 0968-1302
PUBLISHER: Arnold, Hodder Headline
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English

AB A review with 56 refs. Conventional nonsteroidal anti-inflammatory drugs

(NSAIDs) inhibit both of the cyclooxygenase (COX-1, and COX-2) enzymes to varying degrees; consequently, they impair prostaglandin prodn. in all tissues, causing adverse effects, esp. in the gastrointestinal tract, respiratory system (aspirin-induced **asthma**), kidney and haematol. system. Unfortunately, side-effects are common when these nonselective NSAIDs are given and many patients have contraindications to their use. The anti-inflammatory actions of the NSAIDs are mediated by **COX-2 inhibition**, while the adverse effects are considered to be predominantly caused by COX-1 inhibition. Therefore, the selective inhibition of **COX-2 inhibitors** offers real hope for safer NSAIDs; specific agents have now been developed to do this. Selective **COX-2 inhibitors** are effective anti-inflammatory agents, but their analgesic efficacy is still unclear. While they have significantly less gastrointestinal and antiplatelet effects, the acute renal and pulmonary effects of selective **COX-2 inhibitors** have not been fully clarified. Moreover, there are issues concerning their long-term safety because the inhibition of constitutive COX-2, which appears to have some important physiol. functions, may still cause adverse effects.

REFERENCE COUNT: 56 THERE ARE 56 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 12 OF 12 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 1999:396789 HCAPLUS
DOCUMENT NUMBER: 131:210723
TITLE: New highly selective **COX-2 inhibitors**
AUTHOR(S): Ford-Hutchinson, A. W.
CORPORATE SOURCE: Merck Frosst Centre for Therapeutic Research, Kirkland, QC, H9H 3L1, Can.
SOURCE: Selective COX-2 Inhibitors: Pharmacology, Clinical Effects and Therapeutic Potential, Proceedings of a Conference, Cannes, Fr., Mar. 20-21, 1997 (1998), Meeting Date 1997, 117-125. Editor(s): Vane, John R.; Botting, Jack H. Kluwer: Dordrecht, Neth.
CODEN: 67UBAO
DOCUMENT TYPE: Conference; **General Review**
LANGUAGE: English

AB A review, with 55 refs. Cyclooxygenase (prostaglandin G/H-synthase, COX) exists in two isoforms which have been termed COX-1 (constitutive enzyme) and COX-2 (an inducible enzyme). The most significant differences between COX-2 and COX-I are in their regulation as COX-2 can be induced transiently over a >50 fold range by a variety of inflammatory mediators as well as stimuli such as hypoxia, synaptic excitation, injury and laminar sheer stress, and simply by incubation of tissues in vitro. The mechanism of action of non-steroidal anti-inflammatory drugs involves inhibition of COX. In addn., inhibition of the prodn. of prostaglandins (PGs) explains the anti-inflammatory, analgesic and anti-pyretic activity of these compds. as well as their ability to inhibit hormone-induced uterine contractions and certain types of cancer growth. It is also abundantly clear that non-steroid anti-inflammatory drugs (NSAIDs) have mechanism-based side effects which include induction of gastrointestinal lesions, effects on renal function in compromised individuals, increases in bleeding time, induction of NSAID-induced **asthma** and prolongation of gestation and labor. Thus, it is clear that prostanoids have both physiol. and pathol. effects. The hypothesis behind the development of selective **COX-2 inhibitors** is that the therapeutic usefulness of NSAIDs will be largely due to inhibition of inducible COX-2, while the side effect profile will be mainly due to inhibition of COX-I. All the NSAIDs currently on the market in North America show no significant degree

of selectivity for COX-2. Preclin. and early clin. data supports the hypothesis that selective **COX-2 inhibitors** will have anti-inflammatory, analgesic and anti-pyretic activities comparable to NSAIDs with a substantial redn. in some of the side effects assocd. with this class of drugs, particularly induction of gastric lesions and effects on bleeding times. The effects of selective **COX-2 inhibitors** on renal function in renally-compromised individuals remains to be detd. Mechanistic studies indicate that a high degree of in vitro biochem. selectivity for COX-2 will be required in order to achieve effective functional selectivity in vivo.

REFERENCE COUNT: 55 THERE ARE 55 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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     (FILE 'HOME' ENTERED AT 10:48:57 ON 15 JUN 2004)

     FILE 'REGISTRY' ENTERED AT 10:49:17 ON 15 JUN 2004

     FILE 'HCAPLUS' ENTERED AT 10:49:39 ON 15 JUN 2004
L1          2615 S COX-2 () INHIBIT?
L2          1536 S L1 AND INFLAMMAT?
L3           434 S L2 AND REVIEW/DT
L4            22 S L1 AND INFLAMMAT? () DISORDER?
L5             6 S L4 AND REVIEW/DT
L6            41 S L1 AND ULCERAT? () COLIT?
L7             1 S L6 AND REVIEW/DT
L8            72 S L1 AND ASTHMA?
L9            12 S L8 AND REVIEW/DT

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          5134 CROHN?
L10         56 L1 AND CROHN?

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       1734424 REVIEW/DT
L11           1 L10 AND REVIEW/DT

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L11  ANSWER 1 OF 1  HCAPLUS  COPYRIGHT 2004 ACS on STN
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Full Text | Citing References

ACCESSION NUMBER: 2002:661516 HCAPLUS
DOCUMENT NUMBER: 138:378294
TITLE: Nonsteroidal anti-inflammatory drugs and inflammatory bowel disease: current perspectives
AUTHOR(S): Cipolla, Giovanna; Crema, Francesca; Sacco, Stefano; Moro, Elisabetta; De Ponti, Fabrizio; Frigo, Gianmario
CORPORATE SOURCE: Department of Internal Medicine and Therapeutics, University of Pavia, Pavia, 27100, Italy
SOURCE: Pharmacological Research (2002), 46(1), 1-6
CODEN: PHMREP; ISSN: 1043-6618
PUBLISHER: Elsevier Science Ltd.
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English

AB A review. Mechanisms underlying the gastric toxicity of nonsteroidal anti-inflammatory drugs (NSAIDs) have been extensively investigated, whereas those leading to intestinal damage are not completely understood.

Several hypotheses have been put forward on the pathophysiol. of intestinal damage by NSAIDs: enhanced intestinal permeability, inhibition of cyclooxygenase (COX), enterohepatic recirculation, and formation of adducts. The effects of COX-2 selective inhibitors, which appear to have better gastric tolerability when compared to nonselective NSAIDs, on normal and inflamed intestinal mucosa (as in **Crohn**'s disease or ulcerative colitis) are still largely unexplored. If **COX-2 inhibition** plays a key role in suppressing the inflammatory process, recent evidence suggests that COX-2 products are involved in maintaining the integrity of intestinal mucosa, in the healing of gastrointestinal ulcers and in the modulation of inflammatory bowel disease (IBD). Animal models of intestinal inflammation have so far yielded conflicting results on the effects of COX-2 selective inhibitors on the intestinal mucosa. It is now clear that NSAIDs do not act through cyclooxygenase inhibition, but also have different targets such as nuclear factor-κB and/or peroxisome proliferator-activated receptors γ. The peculiar pharmacol. profile of each compd. may help to explain the different impact of each NSAID on the inflammatory process and on IBD. Notably, the salicylic acid deriv. 5-ASA is widely used in the treatment of IBD and is believed to act through nuclear factor-κB inhibition. Although the use of COX-2 selective inhibitors remains contraindicated in patients with IBD, studying their effects on intestinal mucosa may offer new insights into their subcellulars mechanisms of action and open new avenues for the development of novel therapies for IBD.

REFERENCE COUNT: 56 THERE ARE 56 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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     (FILE 'HOME' ENTERED AT 10:48:57 ON 15 JUN 2004)

     FILE 'REGISTRY' ENTERED AT 10:49:17 ON 15 JUN 2004

     FILE 'HCAPLUS' ENTERED AT 10:49:39 ON 15 JUN 2004
L1          2615 S COX-2 () INHIBIT?
L2          1536 S L1 AND INFLAMMAT?
L3           434 S L2 AND REVIEW/DT
L4            22 S L1 AND INFLAMMAT? () DISORDER?
L5             6 S L4 AND REVIEW/DT
L6            41 S L1 AND ULCERAT? () COLIT?
L7             1 S L6 AND REVIEW/DT
L8            72 S L1 AND ASTHMA?
L9            12 S L8 AND REVIEW/DT
L10           56 S L1 AND CROHN?
L11            1 S L10 AND REVIEW/DT

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          4271 GASTRIT?
L12         33 L1 AND GASTRIT?

=> s l12 and review/dt
       1734424 REVIEW/DT
L13          5 L12 AND REVIEW/DT

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Full Text | Citing References

ACCESSION NUMBER: 2003:919785 HCAPLUS
DOCUMENT NUMBER: 139:374127
TITLE: Balancing gastroprotection and cardioprotection with selective cyclo-oxygenase-2 inhibitors: Clinical implications
AUTHOR(S): Meagher, Emma A.
CORPORATE SOURCE: Department of Medicine and Center for Experimental Therapeutics, University of Pennsylvania School of Medicine, Philadelphia, USA
SOURCE: Drug Safety (2003), 26(13), 913-924
CODEN: DRSAEA; ISSN: 0114-5916
PUBLISHER: Adis International Ltd.
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English

AB A review. NSAIDs have been the mainstay of treatment in the management of pain and inflammation assocd. with chronic inflammatory disorders. They are effective. However, complications arising from chronic NSAID use are common and are primarily due to gastrointestinal (GI) toxicity in the form of **gastritis**, peptic erosions and ulceration and GI bleeds. GI toxicity has been attributed to the blockade of the cyclo-oxygenase (COX)-1-mediated generation of the cytoprotective prostanoids, such as prostaglandin (PG) E2 and PGI2 (prostacyclin). More recently, selective **COX-2 inhibitors** ('coxibs') were designed to inhibit the prodn. of COX-2-dependent inflammatory prostanoids and to leave intact the cytoprotective COX-1 products. The coxibs, while exhibiting similar efficacy to traditional NSAIDs in controlled clin. trials of their efficacy in chronic inflammatory conditions, such as osteoarthritis and rheumatoid arthritis, have been assocd. with a reduced incidence of surrogate or actual indexes of GI toxicity. However, concerns regarding cardiovascular safety in high-risk patients have evolved. These concerns were driven initially by the concept that inhibition of COX-2-derived endothelial PGI2 without concomitant inhibition of platelet thromboxane A2 would result in increased cardiovascular risk. This was borne out in the Vioxx Gastrointestinal Outcomes Research study of rofecoxib, but not demonstrated in the Celecoxib Long Term Arthritis Safety Study trial. Further elucidation of the relative roles of COX-1- and COX-2-generated prostanoids has enabled a greater understanding of the biol. of these pathways. However, it is still not completely clear how this understanding may be appropriately translated into clin. medicine.

REFERENCE COUNT: 70 THERE ARE 70 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 2 OF 5 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2003:749509 HCAPLUS
DOCUMENT NUMBER: 140:104272
TITLE: **COX-2 inhibition**, H. pylori infection and the risk of gastrointestinal complications
AUTHOR(S): Chan, Francis K. L.
CORPORATE SOURCE: Department of Medicine & Therapeutics, Prince of Wales Hospital, Hong Kong, Peop. Rep. China
SOURCE: Current Pharmaceutical Design (2003), 9(27), 2213-2219
CODEN: CPDEFP; ISSN: 1381-6128
PUBLISHER: Bentham Science Publishers Ltd.
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English

AB A review. Current data on the gastric safety of cyclooxygenase-2